REPORT OF CASE OF EXCEEDINGLY RAPID AND VERY SLOW RESPIRATION, WITH PAUSES IN RESPIRATION VARYING FROM TWENTY SECONDS TO TWO MINUTES IN DURATION IN A PATIENT SUFFERING FROM TUBERCULOUS MENINGITIS, SYPHILITIC PERIARTERITIS OF THE PONS AND MEDULLA AND FROM HYSTERIA.[1]

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Mrs. B., white, *aet.* 29, widow, born in Kansas, teacher by occupation, was first seen by me in consultation with Dr. Brasher, November 3, 1899.

Family history.—Father died of cancer of the bowel and stomach; mother of consumption; other members of the family suffered from lung trouble; two of mother's sisters died from pulmonary tuberculosis.

Personal history.—She was well and strong in childhood. She had scarlet fever at her fifteenth year, but seemed to recover perfectly. She began to menstruate at her fourteenth year, and was comparatively well up to the time of her marriage, at nineteen years of age.

Six months after her marriage she was in a railroad wreck. She was thrown forward with considerable violence and the top of her head struck the upper edge of the seat in front of her. Immediately after she complained of great pain in the posterior portion of her head and in the cervical and upper dorsal region of the spine. She was confined to her bed for a period of three months, suffering from nervousness, apprehension, great prostration, headache, and developed general hysterical symptoms. During the paroxysms she would throw herself from one side of the bed to the other, scream and pull

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(The most rapid respiration was 140 to 142 per minute, the slowest from 2 to 3 per minute, the latter extending over prolonged periods.)

[1] Read before the Rocky Mountain Interstate Medical Association at its Annual Session, September 3 and 4, 1901.

her hair with both hands. At these times she had repeated convulsions, which, evidently, were hysterical in character, as she assumed an opisthotonic position, with body arched forward, so that she threw her entire weight on her feet and back of head. These paroxysms were short in duration, but recurred repeatedly for several months, and occasionally up to the time of the development of her fatal illness and were present during the early portion of this. It was thought that she had had one or two miscarriages two or three years after her marriage, but I could not learn anything definite about these, because I saw her too late to get a reliable history from the patient. During four or five years immediately succeeding her marriage she had lived in Oregon with her husband several hundred miles from her family and most intimate friends.

Six years after her marriage she gave birth to a syphilitic child. It had all the ear-marks of syphilis and lived only a few weeks after birth. Her husband was known to be infected from syphilis.

Soon after the death of her child she separated from her husband, returned to Denver and lived with her relatives a while. During her stay in Denver, Dr. Brasher, who was the family physician, saw her and detected some tubercular trouble in the apex of the left lung. She improved and was able to support herself by teaching school, although she occasionally, especially when overworked or worried, would have a slight hysterical paroxysm.

After residing six weeks in Cripple Creek, at an elevation of about 9,000 feet, on October 25, 1899, she was seized, while sitting in her chair, "with a fainting spell," and this was followed, almost immediately, by a hysterical paroxysm. This attack was relieved by a hypodermatic injection of morphine, but as soon as the medicine began to lose its effects, she became hysterical, complained bitterly of headache, great weakness, a smothering sensation, and if not relieved by morphine, would pass into an apparent hysterical paroxysm, during which she would scream, pull her hair, arch her body forward and throw herself from one side of the bed to the other. It was stated that her body and limbs assumed all kinds of positions seen in the major attacks of hysterical paroxysms. I have been unable to learn whether her pulse, temperature and respiration were observed.

On November 1, 1899, her sister brought her to Denver and placed her under the care of Dr. Brasher, with whom I saw her two days later.

Examination, November 3, 1899.—The patient is lying in bed moaning, holding her head with both hands and complaining of heachache. She is pale, anemic and evidently has lost considerable flesh, to which her sister, who had been with her in Cripple Creek, testifies. She is nervous and manifests many hysterical symptoms. Dr. Brasher tells me that he had been able to give her relief from headache and secure sleep only by the administration of morphine hypodermically. He corroborates the statement of her relatives that she is taking but little food and that she vomits most of that which she takes. I am informed that even a few teaspoonfuls of water make her vomit.

No paralysis or special weakness of any group of muscles is found. The deep reflexes are increased, but there is no ankle-clonus. The tendo-Achillis reflexes are well marked. The superficial reflexes are present. The plantar reflexes are normal in character. No loss of general sensation is detected. The organs of the special senses appear quite acute, with the exception of those of vision. Here the fields of vision are narrowed and the acuity of vision is lessened. It is difficult accurately to test vision on account of the severe headache from which she seems to be suffering. She is conscious, but her answers to questions do not seem to be always reliable. The optic discs are pale, but not atrophied. The pupils are equal and respond well to light and accommodation. She had not, at any time, complained of diplopia, and none is found at the time of my examination. The temperature at 11.30 A.M. is 100° F.; pulse 120; respiration 24, and otherwise normal in character. Dr. Brasher states that the temperature during the two days that he had attended her had varied from normal or below to 102° F.

I express the opinion, that while many of the patient's symptoms are hysterical in character, she is suffering from some obscure disease of the brain. Its exact nature I am unable to determine.

The heart is free from disease, the apex of the left lung is partially consolidated, but the other viscera show no evidence of disease.

A course of treatment, in which codeine is substituted for morphine, when an anodyne is absolutely necessary, is outlined.

Five days later Dr. Brasher reported to me that the patient seemed to be getting worse, he could not get along without morphine and requested me to put the patient in St. Luke's Hospital and take sole charge of her.

She is admitted into the hospital November 8, 1899, at 3 P.M. She is complaining of headache, pain in the abdomen and constant nausea. Appetite is poor and she vomits occasionally even after taking only water. She is restless, moans a great deal and seems very miserable. At 4 P.M. T. 100.4° F.; P. 92; R. 24. The temperature by 6 P.M. is slightly below 99° F. and remains so until 3.45 A.M., of the 9th, when it, while she is sleeping, rises to 100.8° F.; pulse and respiration remaining about the same as recorded one hour after her admission into the ward. At 9 A.M. and 12 M. of the 9th temperature is 102° F.; pulse 104; respiration 24.

The patient is exceedingly weak, has lost some flesh since I had first seen her in consultation several days previously. The character of the pulse is not good. It is irritable, poorly sustained and the volume is small. I detect nothing in the character of the respiration to arrest attention.

A careful examination reveals nothing beyond what I had observed at my first visit before the patient had been brought to the hospital. It is reported that the patient was sleeping very poorly before she was brought to the hospital, but the first night in the hospital she slept six hours without an anodyne or a hypnotic. While she is still hysterical she has shown no violent outbreaks like those that had been observed while she was at home. The urine contains neither albumin nor sugar. An examination of the lungs reveals no active trouble. There is some dulness over the left apex and a little prolonged expiration in this portion, but there are no râles of any kind. There is neither cough nor expectoration. Neither the liver nor spleen is enlarged.

The irregular character of the temperature made me apprehensive lest there might be some organic brain trouble. Its tendency to be higher in the morning than in the afternoon determines me to try methylene blue lest she may be suffering from chronic malaria. She had lived in a malarial district and her head pain is said to be worse during the latter part of the night. The medicine after several days' trial has no appreciable effect. On account of pain in the lower portion of the abdomen, Dr. W. A. Jayne is requested to make an examination of the uterus and its appendages. He finds no inflammation, no ovarian trouble, nothing beyond a displacement of the womb.

November 10, 9 P.M., T. 103° F.; P. 104; R. 24.

November 10, 10 P.M., T. 103.7° F.; P. 108; R. varies from 10 to 22 per minute. She is in a stuporous condition. The temperature is taken in the left axilla. There are distinct intermissions of respiration while the patient is asleep. Breathing is more rapid while the patient is asleep than while she is awake. Both optic nerves appear more congested than normal and there is venous engorgement of the discs. The condition may be termed a passive hyperemia. The reflex from the lymphatics of the disks and fundi is quite marked. She has complained once or twice during the day of not being able to see for a short time. She is taking a capsule to relieve pain, containing phenacetine 2 grs., caffeine 1 gr., extract cannabis indica (Hering's) 1-6 gr., codeine, 1-4 gr., monobromate of camphore, 2 gr., given every two or three hours if necessary. Ten grs. sodium bromide are ordered to be administered every three hours, while she is restless. She receives no morphine after she enters the hospital.

On account of the history of syphilis she is given inunctions of mercurial ointment thrice daily and potassium iodide is administered internally.

At 12 o'clock, midnight, the temperature has dropped to 100.4° F.; pulse to 98 and respiration to 14. Early in the morning of the 11th the temperature is about normal. At 8 A.M. of the same morning I find respiration rapid and irregular in frequency and character. It varies from 36 to 38 per minute. She is inclined to fall asleep while I am sitting by her bed. The respiration increases in frequency as soon as the patient falls asleep. The temperature rises during the morning and registers 102.4° F. at 12 M., and drops one or two degrees during the latter part of the day.

Nov. 12. Projectile vomiting on awakening in the early morning. Respiration 26 while awake, 32 while asleep. Temperature at 9.30 p.m. is 103° F. It is observed during the early part of the evening that respiration is irregular at times. Some respiratory acts are much longer than others. This occurs only while she is asleep. The disks present a condition of hyperemia, with slight obscuring of the edges of the disks, especially on the temporal sides. She is nauseated at times during the day and occasionally vomits.

Nov. 13. Complains of pain in post cervical muscles. Slight optic neuritis is apparent. Respiration varies from 13 to 40 per minute during the day; temperature from 102° to 104° F., highest at 6 A.M.; the pulse varies but little. At noon I note that the breathing varies from to 13 to 34 during the few minutes that I spend in watching her. The rapid respiration and the distinct intermissions invariably occur while the patient is asleep. Respiration is becoming "up and down," or "ascending and descending" in character without the intermissions seen in Cheyne-Stokes breathing.

Nov. 14. Respiration intermittent in character only while asleep, 29 respirations in 3 minutes. Intermissions 25 seconds in length occur. During the intermissions the patient sleeps calmly, but as soon as respiration is resumed the muscles of the face, especially around the nose and over the forehead, contract, evidently on account of pain. Each respiratory act following a prolonged intermission is accompanied by a sigh, or, it is more properly termed, a groan of distress. Legs are flexed at knees and thighs at hips.

Nov. 15. Patient is in a stupor and lies with mouth open; temperature variable, pulse and respiration increasing in frequency. The legs are no longer flexed at knees and hips.

The next few days witness but little change except the temperature is lower, the pulse less frequent and poorer in quality, and breathing is becoming more rapid. From the 18th to the 20th respiration is about 50 per minute. She is unconscious for 4 or 5 days. Double optic neuritis without much swelling of the disks. Slight hemorrhagic extravasation on the nasal side of right disk and on the temporal side of the left.

Nov. 21. Eight A.M. T. 100° F.; P. 124; R. 44; respiration is irregular and very shallow. If not aroused she lies in a stuporous condition. She lies with eyes open wide; there is almost constant nystagmus, occasional twitching of face muscles and constant delirium while awake. Respiration while awake varies from 10 to 16 per minute, while asleep it is 40 and decidedly intermittent and very shallow. Pulse 135. She is troubled a great deal with hiccough.

On Nov. 22, temperature about normal, pulse 110; respiration 10 to 11 per minute while awake, 34 while asleep.

Nov. 26, little change to note in patient's condition since the 22nd. At 8 A.M. a chill occurs, lasting 20 minutes, pulse becomes imperceptible, respiration 9 per minute. She vomits a great deal during the day. No rise in temperature during or following the chill.

Nov. 28. Temperature about normal, pulse 66, respiration varies from 5 to 11 per minute for nearly 36 hours. Several intermissions in respiration varying in length from 1 1-2 to 2 minutes occur. I observe intermission of 90 seconds in duration. The nurse, who is very accurate, registers several intermissions of 2 minutes in length during the night in which no attempt at breathing could be detected. Death seems imminent for three days during which it is almost impossible to get her to swallow any nourishment. At one time while I was present she ceases to breath for a period of 110 seconds, her face becomes cyanosed, and her whole body rigid. On adopting artificial respiration she begins to breath and the muscles relax.

During the next two or three days respiration remains very slow, then it begins to increase in frequency and on December 3 varies from 13 to 66 per minute. No change in temperature or pulse. December 4, temperature low, 95.8° F.; pulse rapid, 128; respiration 18 to 58.

Dec. 6, respiration 82; P. 130; T. 101° F.; patient is unconscious, and has been so most of the time since November 28.

Dec. 13, pulse and temperature about the same, but respiration from 54 to 110; most of time during 24 hours it is above 80.

Dec. 14, respiration reached 142 per minute. I count it when it is 130. Patient unconscious. Both optic nerves white. The tabular view of the respiration is exceedingly interesting for the next few days or weeks.

Dec. 18, post-cervical muscles quite rigid and head retracted slightly.

Dec. 26, head greatly retracted, pulse very difficult to count, it is small, weak and varies from 130 to 150 per minute.

Jan. 4, respiration 138.

Jan. 9, death occurs at 8.45 P.M. Respiration remains rapid, varying from 50 to 100 or more until a few hours before death. During the last four hours of life respiration varies from 24 to 30. Fifteen minutes before death it is 24. The temperature one and three-quarters hours before death is 104.2° F.; 45 minutes before death 102.6° F.; at death 100.4° F. About six weeks before death two bedsores began to form, one just to each side of the lower portion of the spine. These proved rebellious to care and treatment and great sloughs formed. There was little more than skin, bones and tendons left at the time of death, so extreme had emaciation become.

Autopsy, 15 hours after death. Permission was obtained to examine the brain only. Post-mortem rigidity was well marked.

Nearly all the adipose tissue had disappeared from the scalp; no abnormal adhesions of the dura to the bone. Over the entire convex surface of the brain the membranes and cortical substance of the brain appeared free from any pathologic change. The membranes were easily detached without tearing the cortex. The brain was removed intact, without difficulty, from the lower cavity of the skull. The lateral surface of the brain presented nothing abnormal, except at the fissures of Sylvius. Here the temporo-sphenoidal lobes are separated with great difficulty from the frontal lobes on account of thickening of the membranes and the adhesions that had formed between them and the brain cortex. The pia at these points could not be detached without tearing the cortex. On exposing the upper portions of the fissure of Sylvius, they were found the seat of inflammation and the blood vessels were engorged. The pia was greatly thickened, adherent to the cortex, and every little vessel was distended with blood. Along these portions of the middle cerebral arteries numerous tubercles and small tubercular nodules were found.

On laying the brain on its convex surface and exposing the basilar surface, the lower portions of the fissures of Sylvius presented a condition similar to that found in their upper parts, including the deposition of tubercles along the vessels. There was only slight congestion along the anterior cerebral arteries, the tubercles were few and the thickening of the pia was slight. Over the optic chiasm and surrounding portions, the pia was greatly thickened and firmly adherent to the brain and chiasm. This evidently accounted for the optic neuritis and later for the atrophy of the optic nerves. Along the posterior cerebral arteries as they wind around the pons, especially on its posterior surface, the structures were inflamed, numerous tubercles were found and the pia was thickened and adherent to the brain. Both lateral ventricles contained considerable opaque, turbid-looking fluid in which there was found a quantity of floculent substance, probably lymph. In each posterior horn, more abundant in the right than in the left, there was a deposit of yellowish lymph or pus. It had the appearance of pus, but was free from any odor. The lateral ventricles were considerably distended, the fourth ventricle and the iter were slightly enlarged.

On making the usual incisions into the cerebral and cerebellar substance the parts were normal in appearance, except that they were nearly bloodless. The pons and medulla were cut into blocks, but the blocks were not entirely detached from each other, so as not destroy the relations between any of the parts. These were placed in a one per cent. solution of formaldehyde and given to Dr. Wm. M. Mitchell, together with the fluid from the lateral ventricles for microscopic examination.

REPORT OF DR. MITCHELL, FEBRUARY 13, 1900.

About 35 c. c. of fluid from the lateral ventricles were centrifuged and the sediment examined, both stained and unstained. No tubercle bacilli could be demonstrated. The sediment was found to consist of red blood cells, pus cells, fibrin and granular detritus.

The medulla oblongata and pons were hardened in weak solution of formaldehyde and when sufficiently prepared, sections were made and stained with various reagents.

Particular attention was paid to the sections which were made through the region of the respiratory center. Here it was found that there were areas of round-celled infiltration which were confined principally to the capillaries and smaller arteries. These areas were particularly numerous in the region of the floor of the fourth ventricle. When a small vessel was cut lengthwise, as there were several running lengthwise along the floor of the ventricle, the infiltration was plainly visible extending along the entire length of the vessel, and the uniformity of the cellular growth gave the arteries a heavy, inflamed appearance. When a small vessel was severed crosswise, the round cell growth could be plainly seen investing the vessel and extending into the surounding tissue.

No giant cells were visible and no areas of softening were discovered.

The sections gave typical pictures of what I take to be small or miliary gummata, or periarteritis.

Before attempting an explanation of some of the remarkable symptoms of this case I will give a short résumé of the most important features of the clinical history.

From November 8 to November 27, respiration varied from 10 to 60 per minute, but it usually was about 24. The pulse ranged from 90 to 128; the temperature from 98 to 103° F.

For about 36 hours following the 27th respiration varied from 2 to 11 per minute, but usually it was about 4 or 5, with, at times, complete intermissions in respiration, lasting from 40 seconds to 2 minutes. The pulse during this time varied from 90 to 162; the temperature was slightly below normal.

The next 24 hours, the respiration varied from 9 to 16 per minute, the pulse from 90 to 162; temperature was subnormal.

For 24 or 36 hours following the last period just noted, respiration ranged from 2 to 18 per minute, but it usually was exceedingly slow, pulse 100 to 130; temperature slightly above normal.

The first 12 days of December respiration varied from 4 to 80 per minute; pulse averaged about 80, but sometimes reached 130; temperature 99 to 100° F. There seemed to be no relation between pulse and respiration.

From December 12 to 19, respiration occurred from 42 to 142 per minute and averaged over 90. The respiration at times was more rapid than the pulse. Temperature and pulse were about the same as noted during the first twelve days of December until the latter part of this period, in which the pulse began to increase in frequency, and the body heat to rise and respiration to lesson in number per minute.

From December 19 to January 9, when death took place, the respiration varied from 8 to 138. It usually was rapid, ranging from 40 to 80 or 90 per minute. The pulse most of the time was rapid, but about as fast when the respiration was slow as it was when the respiration was quick. It was noted throughout the progress of the case the slight, or almost imperceptible, influence codeine had on modifying the rapidity of respiration. The temperature continued above normal most of the time during the last three weeks of life.

Seventeen days before death the patient became conscious, rallied and was able to converse rationally nearly all day.

Nine days before death, she again became conscious, but was unable to speak so as to be heard. She seemed entirely blind.

It was observed repeatedly during her unconscious periods, sometimes lasting several days or a week, that the frequency of respiration would vary from 28 to 68 within two minutes. These variations occurred without her moving or any one disturbing her. It should be noted that extremely rapid or slow respiration never occurred while the patient was fully conscious.

It should be remembered that intermissions in respiration first occurred while the patient was asleep and were always most marked at this time, or during the periods when she was profoundly unconscious. The respiration was most rapid, or slowest, when it varied from the normal, while the patient was asleep, or unconscious from other causes. Extreme rapidity of respiration, 100 to 142 per minute, or extreme slowness of respiration, from 2 to 4 per minute, only occurred during periods of profound unconsciousness and never during normal sleep.

Pathology.—So far as I have been able to learn none of Mrs. B.'s relatives have been hysterical. She was not hysterical until after she was injured in a railroad wreck when she was nineteen and one-half years of age. Just the exact nature of the injury no one will ever know, but the patient complained bitterly of pain in the back of the head, in the upper portion of spine, almost immediately after her body had been thrown forward with considerable violence, the top of her head striking on the upper edge of the car seat in front of her. Soon after this accident she manifested hysterical symptoms and was confined to her bed for a period of three months. She remained hysterical ten years, or the remainder of her life. In her case I think we are justified in presuming that she sustained at the time of the wreck some organic lesion in the medulla, pons or adjacent parts, and that the subsequent hysteria was the screen by which the true state of affairs was hidden from view, or, as Dr. S. Weir Mitchell has expressed it, "Hysteria was painted on an organic background."

We know that injuries to the brain determine to a certain extent, the location of syphilitic lesions of this organ. It is probable that the old injury to the medulla rendered the arterioles in this locality more vulnerable to the syphilitic poison than those of other portions of the nervous system. To what extent the tubercular meningitis modified the symptoms of the lesion in the respiratory region of the medulla, it is impossible to say.

Points in the diagnosis.—It must be conceded that the problem of diagnosis was a knotty one before the symptoms of organic disease developed sufficiently to render it evident that hysteria was simply the result of organic disease of the brain. The history of an injury to the head and of syphilitic infection many years previously and the presence of tuberculosis of the lungs, together with fever, made it very probable that the patient was suffering from organic disease of the brain. On the other hand we occasionally meet with hysterical subjects in whom we are unable to find any organic disease with fever varying from 100 to 101° F. or more, especially during the latter part of the day. Dr. S. Weir Mitchell and others have reported many such cases. A history of syphilitic infection, or the presence of tuberculosis of the lungs is only corroborative and never positive evidence of organic brain trouble in such cases as the one reported in this paper. The history of hysterical manifestations extending over a period of ten years, the symptoms varying but little in character during these years, pointed strongly to hysteria. Yet there was something about the patient's appearance, especially the marked wasting, that I was informed had taken place within a week, which made me suspicious of organic disease of the brain. I so expressed myself at the time I first saw the patient, but a more definite diagnosis I would not attempt.

After she was placed under my care the first positive sign that I had of organic disease was the respiration being more rapid while the patient was asleep than while she was awake. The irregular character of the temperature, being higher in the early morning than during the latter part of the day, indicated some inflammatory disease of the brain. Unless I had had the services of an excellent and closely observing trained nurse, I should have been kept in the dark in regard to the diagnosis longer than I was.

How are we to explain the very rapid and the extremely slow respiration in this case, together with the phenomenon that there was no apparent relation between the number of respirations and the frequency of the heart's action?

The normal frequency of respiration is about 16 to 24 per minute. The relative frequency of respiration in health to that of the pulse beat is about one to four. In hysteria and in certain diseases, but more especially in those lesions affecting the pneumogastric nerves, or their branches, this relation is lost, and the number of respirations per minute may equal or exceed the number of heart beats per minute.

Dr. S. Weir Mitchell says: "Hysteria breaks all laws, except its own rules of eccentricity."[2] On the same page he gives a tabular view of the pulse and respiration of a hysterical woman whose average frequency of respiration to pulse beats "was about as one to two; and on one day at the same time, respiration was 89 per minute and pulse 88." On page 200 he mentions a case which he saw with two other physicians. The patient had passed into a state of stupor, from which, for two days, it had been impossible to arouse her. The pulse was about 90, respiration was almost imperceptible, but on careful count it was found to be 96 per minute. Dr. Mitchell gave the opinion that the case would, in the end, prove to be hysterical, and he adds, "An opinion justified within a few hours by the repeated occurrence of very violent hystero-epilepsy."

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[2] "Lectures on Disease of the Nervous System, especially in Women." p. 199, edition of 1885.

Dr. Mitchell reports a most remarkable case[8] in the person of a soldier who was shot in the right chest on May 31, 1862, while engaged in the battle of Fair Oaks. The injury resulted in great hyperesthesia of the right lower chest. The ball passed into the lung, and caused bleeding from the lung and was thought to have become encapsulated and remained in the chest cavity. About eight years later, Dr. Mitchell found the patient still suffering from hyperesthesia of the chest, and in addition from very rapid respiration. When the man was quiet in his room, the pulse was 74, respiration 66 per minute. After running up and down one flight of stairs twice, his pulse was 100, respiration 108 to 110 per minute. Ten days later, the respiration and the heart beats were the same per minute, viz., 78. A little exercise caused the heart to beat 95 times per minute and the respiration to increase to 125. The patient was taken before the College of Physicians of Philadelphia, but no one could give an explanation of the remarkable phenomena. I will not tire you with reference to further cases. I have said sufficient to remind you that hysteria is at times attended by exceedingly rapid respiration. Let us study the results of physiological experiments on the pneumogastric nerves before attempting to account for much of the curious conditions observed by Mitchell and some of those observed by me in connection with the case of Mrs. B.

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[8] *Ibid*, pp. 201 to 207.

If both pneumogastric nerves be divided the number of respirations falls in some animals to 2 or 4 per minute. If the central ends of the penumogastric are gently stimulated, the respiration increase in frequency beyond the normal; greater stimulation may slow or arrest respiration. The superior laryngeal nerves, on account of their extreme sensibility, respond to a slighter stimulus than do the pneumogastrics. The superior and inferior laryngeal nerves constitute the inhibitory nerves of respiration.

The subordinate centers of respiration, several in number, are in the brain and spinal cord. The predominating respiratory centers are situated in the medulla. They are two in number and lie behind the superficial origin of the vagi, one on either side of the posterior apex of the calamus scriptorius, between the nuclei of the vagus and accessorius.[4]

The exciting fibers of respiration lie in the pulmonary branches of the vagus, in the optic, auditory, and cutaneous nerves; normally their action overcomes the action of the inhibitory nerves.

It is probable that mild stimulation of the respiratory centers in the medulla will give rise to even more rapid respiration than gentle stimulation of the afferent exciting nerves of respiration. Certainly destructive lesions of the respiratory centers will slow or arrest respiration.

In hysteria the whole sensorium is in a state of excitation, and especially the cutaneous, optic and auditory nerves which increase the frequency of the respirations. Besides in hysteria the power of inhibition is lessened, thus allowing the exciting nerves of respiration or of the heart to act at their own sweet will. We know that exhaustion of the exciting nerves of respiration or of the heart, or of the centers of respiration, or of the heart will cause slowing of respiration or of the heart's action, as the case may be.

May we not in this manner account for the loss of the relative frequency of respiration to the heart's action? It seems to us, at times, that hysteria ignores all laws, while, in fact, the functions of the body are probably just as law-abiding in hysteria as they are in any other disease.

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[4] "Text-Book of Human Physiology," Landis and Sterling, Third Edition, p. 712.

May one not account for the rapid respiration and the loss of the relative frequency of respiration and the heart's action in the case of the soldier, reported by Dr. S. Weir Mitchell in his lectures on nervous disease?[5] In this case while the patient was at rest the number of respirations per minute was slightly less or equalled the frequency of the pulse, and on slight exercise both respiration and pulse were increasd in frequency, but the respiration to a greater extent than that of the pulse; so that, the number of respirations per minute at one time was 125, the pulse was only 95. In this case the cutaneous surface of the right chest (lower) and the right lung were constantly irritated by nerve injuries that would naturally increase the frequency of respiration. Besides, the man had been suffering from a painful injury for a period of eight years, and had undoubtedly become hysterical. In consequence the power of inhibition was at a low ebb. The facial spasm that occurred several times daily would point to irritation of the pneumogastric nerves as these nerves are connected with facial nerves.

In the case that I have given at some length and detail in this paper, the subordinate centers of respiration, the cutaneous, optic and auditory nerves, those of the lungs, cord, and cerebrum were practically held in abeyance by the predominating centers of respiration in the medulla which were directly irritated by a lesion in the medulla. It is probable that the condition of the blood, which ordinarily affects the predominating centers of respiration to a greater or less extent, was overpowered by the lesion in the medulla.

If we were justified in assuming such a condition to have existed, and it seems to me that we are, we can readily see how an irritating lesion in the exciting centers of respiration would increase the number of respirations almost to an unlimited number. On the other hand, an irritating lesion in the inhibitory centers of respiration, might decrease the number of respirations per minute to 1 or 2, or arrest respiration entirely, as occurred on one or two occasions, when the patient seemed dead, and was revived at the end of two minutes by means of artificial respiration.

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[5] See pp. 201 to 207.

In hysterical subjects with exceedingly rapid respiration morphine, codeine, chloral and other drugs that have a depressing influence on the respiratory centers in the medulla, lessen the number of respirations per minute to one-half or one-fourth of what it was before the drug was administered. In the case of Mrs. B. codeine had no such influence on respiration when it was rapid.

In conclusion let me add: (1) That when we find a case of hysteria we perceive the veil that obscures or the cloak that hides the symptoms of organic disease, or gravely disordered functions, of some important organ.

(2) That respiration which is more rapid while the patient is asleep is strong if not positive evidence of organic disease of the brain in the region of the respiratory centers.